

Code No. 11090

**Anti-Human
APP (N) (10D1) Mouse IgG MoAb**Volume : 100 µg

Introduction : Amyloid precursor protein (APP) is precursor protein of Amyloid β which is major constituent of senile plaque in Alzheimer's disease. It is known that there are three major isoforms, APP695, APP751 and APP770, and are generated from alternative splicing of common precursor mRNA. Processing of APP occurs by two major pathways, non-amyloidogenic pathway and amyloidogenic pathway. The non-amyloidogenic pathway is mediated by α and γ -secretases and gives rise to a large fragment known as soluble APP α (sAPP α) and a small 3 kDa peptide known as p3. On the other hand, the Amyloidogenic pathway is mediated by β - and γ -secretases and yields soluble APP β (sAPP β) and Amyloid β . The physiologic function of APP itself is not clear, however, it is supposed that the function of APP in neuron system is different from that in other organ.

Antigen : Recombinant of the N-terminal portion of Human APP

Source : Mouse-Mouse hybridoma
(X63-Ag8.653 \times BDF-1 spleen cells)

Clone : 10D1 **Subclass** : IgG₁

Purification : Purified with Protein A

Form : Lyophilized product from PBS containing 1 % BSA and 0.05 % NaN₃

How to use : 1.0 mL deionized water will be added to the product, then its concentration comes to 100 µg/mL

Stability : Lyophilized product, 5 years at 2 - 8 °C
: Solution, 2 years at -20 °C

Application : This antibody can be used for western blotting in concentration of about 2 µg/mL.
: This antibody can be used for immunoprecipitation in concentration of about 3 µg/test.

Specificity : Not cross-react with mouse.
This antibody can detect all isoforms of APP695, APP751, APP770 and soluble APP (sAPP α and sAPP β).

Reference : Citron M, Oltersdorf T, Haass C, McConlogue L, Hung AY, Seubert P, Vigo-Pelfrey C, Lieberburg I, Selkoe DJ. Mutation of the beta-amyloid precursor protein in familial Alzheimer's disease increases beta-protein production. *Nature* 360 (6405): 672-674 (1992)

Goate A, Chartier-Harlin MC, Mullan M, et al. Segregation of a missense mutation in the amyloid precursor protein gene with familial Alzheimer's disease. *Nature* 349 704-706 (1991)

Selkoe DJ. Normal and abnormal biology of the β -amyloid precursor protein. *Annu. Rev. Neurosci.* 17 489-517 (1994)

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